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Case Report

An autopsy case of internal jugular vein thrombophlebitis involving sepsis following blunt neck injury

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Abstract

We report an unusual case of delayed death due to sepsis following closed blunt injury to the neck. The victim was a 71-year-old male with a clinical history of hypertension, diabetes and gout. He was found dead about three weeks after being assaulted. He had not consulted a hospital after the assault. Forensic autopsy demonstrated a large liquefied subcutaneous hematoma on the right side of the neck, peri- and thrombophlebitis of the right internal jugular vein. Otherwise, there was no evidence of trauma. Histological examination showed dermal vesicles in the skin covering the hematoma, accompanied by marked inflammatory cell infiltration phagocytosing gram-positive streptococci, subcutaneous edema, panphlebitis with partially organized thrombi and bacterial colonies, pulmonary edema and multiple pulmonary microthrombi involving bacterial aggregates. Postmortem serum C-reactive protein and neopterin levels were markedly elevated. These findings suggest sepsis as the cause of death, induced by infected internal jugular vein thrombophlebitis following blunt neck injury involving impaired skin barrier.

Keywords: Blunt injury; Neck; Thrombophlebitis; Sepsis; Neopterin

1. Introduction

Blunt injury to organs in the neck is uncommon,[1,2] due to the anatomic structures of the mandible protecting from these organs major impact trauma,[3] as well as the mobility and compressibility of the neck itself.[4,5] Previous reports have shown that blunt injury to the neck may incidentally cause various complications including syringomyelia, laryngeal fracture, tracheal rupture and carotid artery occlusion.[6,2,7,8] However, secondary infection through the intact skin affecting the cervical vessels appears very unusual. Although vascular inflammation in the neck is infrequent, internal jugular vein thrombophlebitis associated with acute pharyngitis, tonsillitis and parotitis may be accompanied by fatal sepsis (Lemierre's syndrome).[9–14] These complications are closely related to the complicated anatomic structure of the neck organs.

In the present report, we describe an unusual case of delayed death due to internal jugular vein thrombophlebitis involving sepsis following closed blunt neck injury.

2. Case reports

Case history: The victim was a 71-year-old male, who lived alone. The subject had a clinical history of hypertension, diabetes (188 mg/dL), and gout (uric acid 7.7 mg/dL). In October, he told his friend that he had been assaulted by an unknown robber and sustained a blow to the neck. When the friend visited him 14 days later, the subject was lying down at home, but was still alive. Thereafter, he was found dead on his bed on the 20th day. He had not consulted a hospital.

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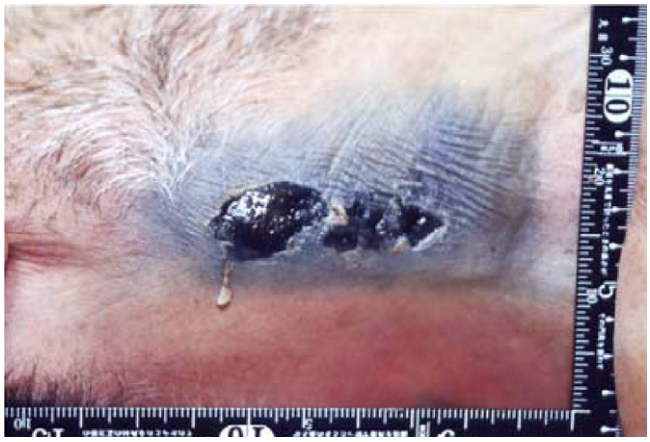

Fig. 1. Right lateral views of the neck, showing dark reddish discoloration and erosions due to vesicle formation.

Autopsy findings: The body was slightly emaciated; 170 cm in height, weighing 51.7 kg. Dark purplish livor was moderately observed on the back, and postmortem rigidity was weak (3–4 days postmortem). The palpebral conjunctivae were pale, and a few petechiae were noted in the right eye. On the right lateral neck region, dark reddish discoloration (5×10 cm) with partial epidermal erosion due to vesicles was observed (Fig. 1). Otherwise, there was no evidence of trauma.

On internal examination, a large liquefied subcutaneous hematoma (15×10 cm) was found in the right neck triangle behind the sternocleidomastoid muscle, covering the right internal jugular vein. The partially organized inner wall of the hematoma adhered to the internal jugular vein, and there was perivascular inflammation, partially organized thrombi in the vascular lumen (Fig. 2). The other neck organs including the right carotid artery were intact. Internal viscera were generally edematous, and petechial hemorrhages were scattered. The heart (460 g) was hypertrophic with enlarged ventricles. There was no evident pathology in the coronary arteries. The lung (left, 445 g; right, 625 g) showed advanced edema. The brain (1375 g) was edematous without any other pathology.

Histological observations: There were dermal vesicles containing gram-positive streptococci in the skin of the neck covering the hematoma, accompanied by marked inflammatory cell infiltration and subcutaneous edema (Fig. 3a). The right internal jugular vein showed partial disruption, dissection and necrosis of the tunica media with bleeding and inflammatory cell infiltration (Fig. 3b), partially organized mixed thrombi involving numerous bacterial aggregates, in which gram-positive streptococci phagocytosed by macrophages were detected. On Azan staining, collagen fiber proliferation in the vascular wall and intravenous thrombi were noted (Fig. 4). The lungs showed edema and multiple microthrombi involving bacterial aggregates (Fig. 5). The pancreas did not show evident fatty infiltration or fibrosis. In the kidney, there was no pathology except for mild thickening of the walls of small arteries. The adrenal glands showed inflammatory cell infiltration.

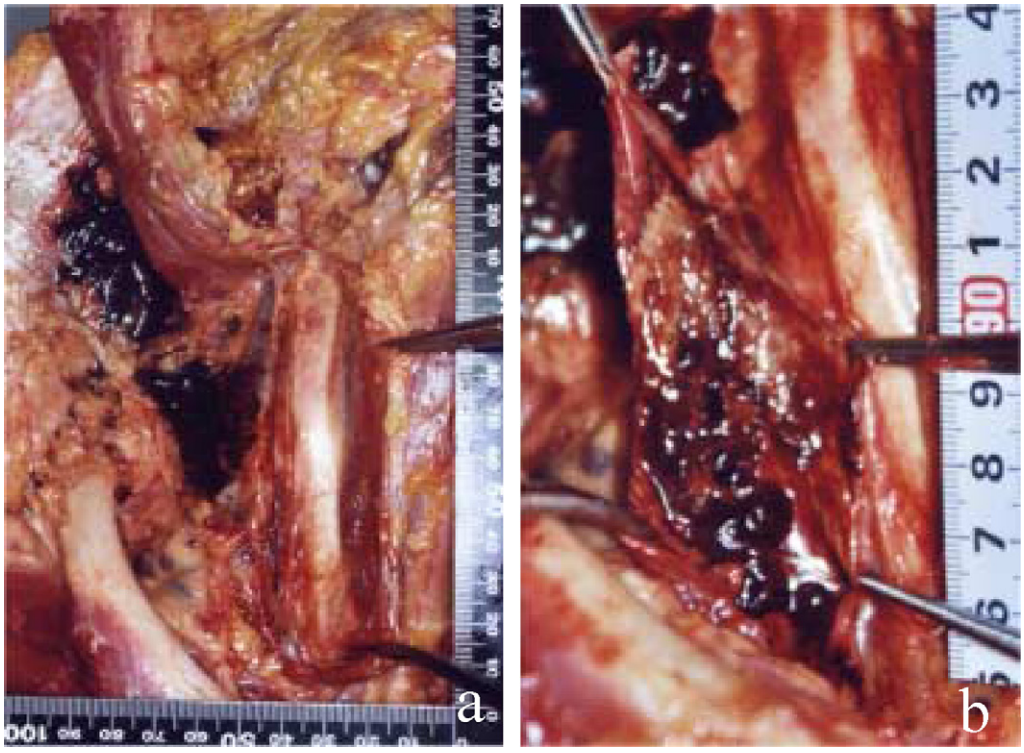

Fig. 2. (a) Internal view of the neck, showing a liquefied hematoma behind the right sternocleidomastoid muscle. (b) Periphlebitis of the right internal jugular vein and partially organized thrombi.

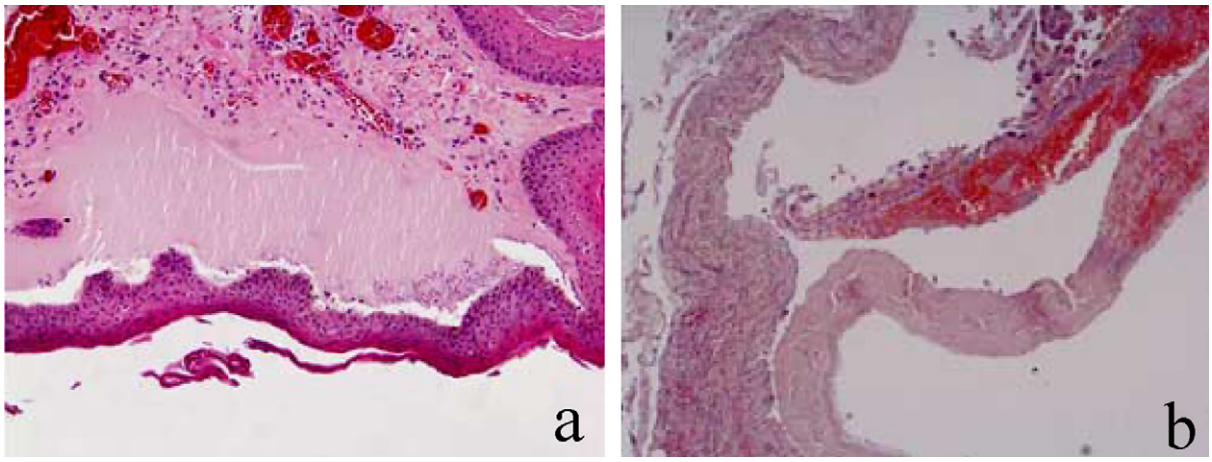

Fig. 3. (a) Histological findings of the skin covering the hematoma. Vesicle formation, bacterial aggregates, and inflammatory cell infiltration were observed (HE staining). (b) Histological findings of the right internal jugular vein. Disruption and dissection of the tunica media with bleeding and inflammatory cell infiltration (HE staining).

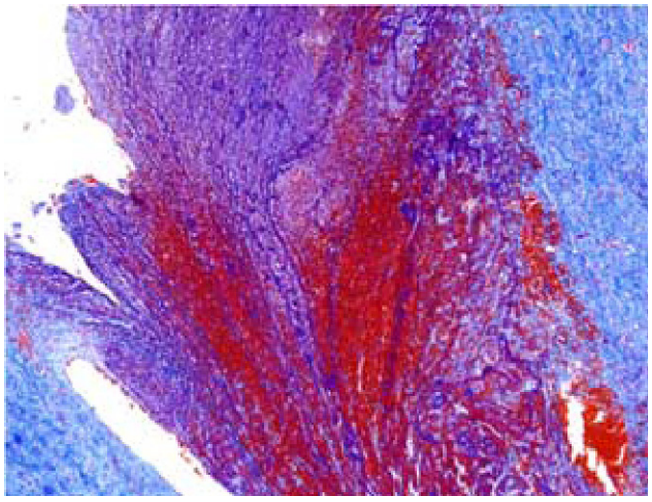

Fig. 4. Collagen fiber proliferation in the vascular wall and thrombi (Azan staining).

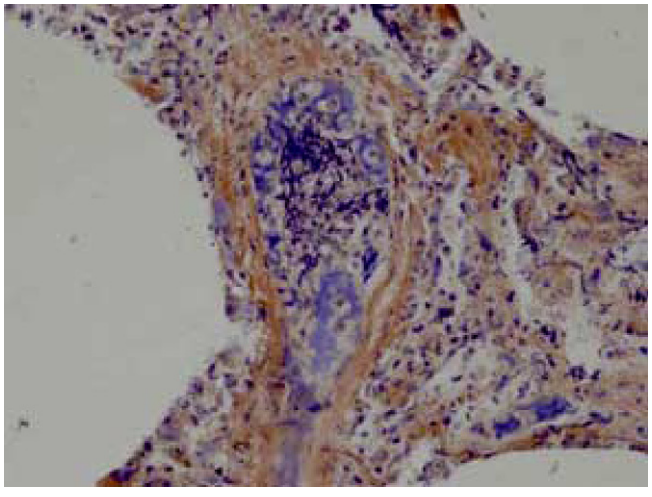

Fig. 5. Histological findings of the lung, showing multiple microthrombi involving bacterial aggregation (PTAH staining).

Postmortem biochemistry: Serum CRP and neopterin were markedly elevated; 28.1 mg/dL (clinical reference value, <0.19 mg/dL) and 995 pmol/mL (clinical reference value, 2–8 pmol/mL),[15,16] respectively. BUN and creatinine were elevated; 111.2 mg/dL (clinical reference value, 6–20 mg/dL) and 4.46 mg/dL (clinical reference value, 0.61–1.04 mg/dL), respectively. Serum acetone was 4.73 μg/mL (clinical reference value, <5 μg/mL). Urine sugar and acetone were negative.

Toxicological findings: Findings on drug screening using Triage® (Biosite Diagnostic Inc., San Diego, CA) were negative. Alcohol was not detected.

3. Discussion

Sepsis is defined as a systemic inflammatory response syndrome in which signs of infection are present.[17] In the present case, systemic inflammatory findings involving bacterial pulmonary microemboli, elevations in serum CRP and neopterin[15,16] strongly suggest sepsis as the immediate cause of death. The primary site of infection causing sepsis was exclusively observed in the neck, showing findings of thrombophlebitis associated with blunt neck injury (contusion) involving dermal vesicles, liquefied hematoma, edema and incomplete disruption of the internal jugular vein. Compared with serum creatinine, marked elevation of BUN was suggestive of the influence of sepsis and large liquefied hematoma of the neck.[18]

Gram-positive streptococci, which were partly phagocytosed, were detected in the skin covering the hematoma, thrombi in the internal jugular vein and pulmonary microthrombi, suggesting bacterial invasion through the skin impaired by blunt injury and subsequent systemic infection. This unusual infection involving thrombophlebitis in closed blunt injury may have been caused by the combined influence of multiple factors related to the anatomic structure of the neck, involving the impaired skin barrier due to

edema, liquefied hematoma, local inflammation and jugular vein injury. These factors may have contributed to vulnerability to bacterial invasion at the site of contusion and the development of thrombophlebitis.[19–22] Partial contribution of the preexisting diabetes may also be considered for the predisposition.[23–25]

Due to the complicated structure of the neck, composed of three layers involving an outer superficial (investing) layer, a middle (visceral) layer and an internal (prevertebral) layer, surgical treatment is difficult; once infection of the neck organs occurs, and the condition may become critical.[5] The prognosis for this infectious complication is poor, with a mortality rate approaching 40% despite aggressive open drainage and antibiotic therapy.[26] However, a recent review of 91 patients with deep-neck space infections, who were diagnosed and treated within 24 h of the initial consultation reported only one death, no septicemia, mediastitinitis, nor jugular vein thrombosis or major vessel disruption.[27] These series suggest the importance of early diagnosis, effective antimicrobial therapy, and adequate surgical drainage.

In the present case, subcutaneous hematoma and venous thrombi showed similar findings of partial organization, which were consistent with the circumstantial evidence of violence 16–17 days before the estimated time of death. There was no evidence of self or social neglect, or medical negligence. The victim may have been unaware of the potential for a fatal outcome. In conclusion, we reported an unusual case of delayed death due to sepsis following closed blunt trauma to the neck, which may be attributed to a peculiar injury involving a large liquefied hematoma and vascular damage based on the characteristic anatomic structure of the neck.

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